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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/080,797	02/21/2002	Romulus Kimbro Brazzell	OP/4-31881A	9942
29180 7590 08/08/2007 BELL, BOYD, & LLOYD LLP P.O. BOX 1135 CHICAGO, IL 60690				
			EXAMINER ANGELL, JON E	
			ART UNIT 1635	PAPER NUMBER
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No. 10/080,797	Applicant(s) BRAZZELL ET AL.	
	Examiner J. Eric Angell	Art Unit 1635	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 22 May 2007.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-3, 8, 27-33, 38-41, 43 and 45-50 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-3, 8, 27-33, 38-41, 43 and 45-50 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

This Action is in response to the communication filed on 5/22/2007.

Claims 1-3, 8, 27-33, 38-41, 43, 45-50 are pending in the application and are addressed herein.

Applicant's arguments are addressed on a per section basis. The text of those sections of Title 35, U.S. Code not included in this Action can be found in a prior Office Action. Any rejections not reiterated in this action have been withdrawn as being obviated by the amendment of the claims and/or applicant's arguments.

Claim Rejections - 35 USC § 102

1. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

2. Claims 1-3, 8, 27, 28, 30, 31, 43, and 45-47 are rejected under 35 U.S.C. 102(b) as being anticipated by Leboulch et al. (WO 99/26480, cited as IDS reference AN), for the reasons of record (e.g., see the Action mailed on 3/14/05), reiterated below for convenience.

As previously indicated, Leboulch teaches a method for treating a human patient suffering from diabetic retinopathy (which can result in corneal, retinal and iris neovascularization) by administering to said patient a nucleic acid molecule which expresses endostatin wherein expression of the endostatin polypeptide in the patient inhibits angiogenesis in the vicinity of the retina (e.g., see claim 33, page 33-34). Specifically, Leboulch teaches that

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the gene therapy vector can be a retroviral vector, adenoviral vector or adenoviral-associated vector (AAV) (see p. 5, lines 19-21). Furthermore, Leboulch indicates that the gene therapy vector can be administered by any method that allows the vector to reach the target cells, such as injection to the target tissue, wherein the target tissue can be the retina of the eye (e.g., see p. 11, lines 10-22; and p. 14, lines 2-14; and claim 33). Therefore, Leboulch anticipates the instant claims.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1 and 29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Leboulch et al. (WO 99/26480, cited as IDS reference AN) in view of Keshet et al. (Journal of Clinical Investigation, 1999) and further in view of Otani et al. (Investigative Ophthalmology & Visual

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Science, 1999) for the reasons of record (e.g., see the Action mailed on 3/14/05), reiterated below for convenience.

As previously indicated, Leboulch teaches a method for treating a human patient suffering from diabetic retinopathy (which can result in corneal, retinal and iris neovascularization) by administering to said patient a nucleic acid molecule which expresses endostatin wherein expression of the endostatin polypeptide in the patient inhibits angiogenesis in the vicinity of the retina (e.g., see claim 33, page 33-34). Specifically, Leboulch teaches that the gene therapy vector can be a retroviral vector, adenoviral vector or adenoviral-associated vector (AAV) (see p. 5, lines 19-21). Furthermore, Leboulch indicates that the gene therapy vector can be administered by any method that allows the vector to reach the target cells, such as injection to the target tissue, wherein the target tissue can be the retina of the eye (e.g., see p. 11, lines 10-22; and p. 14, lines 2-14; and claim 33).

Leboulch does not teach that the method can be used to treat choroidal neovascularization.

Keshet et al. teaches that endostatin is an antiangiogenic peptide that inhibits VEGF activity. Specifically, Keshet et al. teaches, "Endostatin was shown to inhibit VEGF-induced endothelial cell migration in vitro and to have anti-tumor activity in vivo, without any apparent signs of toxicity." (See p. 1500, 1st column, lines 3-6).

Furthermore, it was recognized in the art that vascular endothelial growth factor (VEGF) is involved in choroidal neovascularization (CN). For instance, Otani et al. teaches,

"Recent histological and immunohistochemical studies of experimentally produced and surgically excised CNVMs [choroidal neovascular membranes] have indicated that VEGF, transforming growth factor beta (TGF β), acidic fibroblast growth factor (aFGF) and basic fibroblast growth factor (bFGF) are involved in the mechanism of CNVM

formation associated with ARMD [age-related macular degeneration]. Because VEGF has great selectivity for endothelial cells, it is considered to be a critical angiogenic factor in the development of CVMN, even though the mechanism of CNVM is not fully understood.” (Emphasis added; see paragraph bridging pages 1912-1913).

It is also noted that Otani et al. teaches, “Present findings that Ang2 and VEGF are co-upregulated and that Tie2 is expressed in a variety of cell types in CVNMs further support a crucial role of the interaction between VEGF and Ang2 in pathologic angiogenesis of CNVM formation.” (See p. 1912, Abstract).

Therefore, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to use the method taught by Leboulch to ameliorate or reduce the rate of choroidal neovascularization in a subject with a reasonable expectation of success.

Since the teachings of the prior art indicate that (1) Endostatin is an antiangiogenic factor that inhibits VEGF activity, (2) Endostatin can be used in gene therapy methods to inhibit neovascularization, and (3) VEGF is known to be involved in choroidal neovascularization (e.g., see Otani et al.) one of ordinary skill in the art would have been motivated to use the method of Leboulch to inhibit choroidal neovascularization.

Claims 1 and 32 are rejected under 35 U.S.C. 103(a) as being unpatentable over Leboulch et al. (WO 99/26480, cited as IDS reference AN) in view of US Patent 6,106,826 (Brandt et al.) for the reasons of record (e.g., see the Action mailed on 3/14/05), reiterated below for convenience.

As previously indicated, Leboulch teaches a method for treating a human patient suffering from diabetic retinopathy (which can result in corneal, retinal and iris neovascularization) by administering to said patient a nucleic acid molecule which expresses

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endostatin wherein expression of the endostatin polypeptide in the patient inhibits angiogenesis in the vicinity of the retina (e.g., see claim 33, page 33-34). Specifically, Leboulch teaches that the gene therapy vector can be a retroviral vector, adenoviral vector or adenoviral-associated vector (AAV) (see p. 5, lines 19-21). Furthermore, Leboulch indicates that the gene therapy vector can be administered by any method that allows the vector to reach the target cells, such as injection to the target tissue, wherein the target tissue can be the retina of the eye (e.g., see p. 11, lines 10-22; and p. 14, lines 2-14; and claim 33).

Leboulch does not teach that vector is administered intravitreally.

Brandt teaches gene therapy vectors which can be used to deliver therapeutic genes for gene therapy, and specifically teaches an HSV vector as well as an adenoviral vector and adeno-associated vector for use in gene therapy of the eye wherein the vector can be delivered to the eye by intravitreally injecting the vector as well as subretinally and intraocularly delivering the vector, for therapeutic purposes, such as macular degeneration. (e.g., see abstract, column 5, lines 5-20, column 8, lines 57-65, and column 9 lines 15-20).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time of filing to modify the method taught by Leboulch such that the vector used was delivered intravitreally with a reasonable expectation of success.

The motivation to modify the method of Leboulch is supplied by Brandt who specifically teaches that intravitreal delivery of a therapeutic vector is an effective administration for gene therapy of eye diseases.

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Claims 1, 33 and 38 are rejected under 35 U.S.C. 103(a) as being unpatentable over Leboulch et al. (WO 99/26480, cited as IDS reference AN) in view of US Patent 6,555,107 (Poeschla et al.) for the reasons of record (e.g., see the Action mailed on 3/14/05), reiterated below for convenience.

As previously indicated, Leboulch teaches a method for treating a human patient suffering from diabetic retinopathy (which can result in corneal, retinal and iris neovascularization) by administering to said patient a nucleic acid molecule which expresses endostatin wherein expression of the endostatin polypeptide in the patient inhibits angiogenesis in the vicinity of the retina (e.g., see claim 33, page 33-34). Specifically, Leboulch teaches that the gene therapy vector can be a retroviral vector, adenoviral vector or adenoviral-associated vector (AAV) (see p. 5, lines 19-21). Furthermore, Leboulch indicates that the gene therapy vector can be administered by any method that allows the vector to reach the target cells, such as injection to the target tissue, wherein the target tissue can be the retina of the eye (e.g., see p. 11, lines 10-22; and p. 14, lines 2-14; and claim 33).

Leboulch does not teach that vector is a lentiviral vector or that that the vector is a bovine immunodeficiency viral vector.

Poeschla teaches methods of gene therapy for the eye wherein a lentiviral vector, specifically a bovine immunodeficiency vector, is used to deliver and express the therapeutic gene in the eye of the subject. (For example, see abstract, column 2, lines 10-35; column 4, lines 34-41; column 11, 45-55).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time of filing to modify the teaching of Leboulch such that the gene therapy vector used is the

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bovine immunodeficiency viral vector taught by Poeschla (which is a lentiviral vector) with a reasonable expectation of success.

The motivation to make such a modification is provided by Poeschla. Poeschla teaches that the BIV vector is better for transfecting difficult to target non-dividing cells of the nervous system including eye cells.

Claims 1, 33, 38-41 and 48-50 are rejected under 35 U.S.C. 103(a) as being unpatentable over Leboulch et al. (WO 99/26480, cited as IDS reference AN) in view of US Patent 6,555,107 (Poeschla et al.) and further in view of US Patent 6,106,826 (Brandt et al.) for the reasons of record (e.g., see the Action mailed on 3/14/05), reiterated below for convenience.

As previously indicated, Leboulch teaches a method for treating a human patient suffering from diabetic retinopathy (which can result in corneal, retinal and iris neovascularization) by administering to said patient a nucleic acid molecule which expresses endostatin wherein expression of the endostatin polypeptide in the patient inhibits angiogenesis in the vicinity of the retina (e.g., see claim 33, page 33-34). Specifically, Leboulch teaches that the gene therapy vector can be a retroviral vector, adenoviral vector or adenoviral-associated vector (AAV) (see p. 5, lines 19-21). Furthermore, Leboulch indicates that the gene therapy vector can be administered by any method that allows the vector to reach the target cells, such as injection to the target tissue, wherein the target tissue can be the retina of the eye (e.g., see p. 11, lines 10-22; and p. 14, lines 2-14; and claim 33).

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Leboulch does not teach that vector is a lentiviral vector such as a bovine immunodeficiency viral (BIV) vector or that the lentiviral/BIV vector is administered intraocularly, subretinally or intravitreally.

Poeschla teaches methods of gene therapy for the eye wherein a lentiviral vector, specifically a bovine immunodeficiency vector, is used to deliver and express the therapeutic gene in the eye of the subject. (For example, see abstract, column 2, lines 10-35; column 4, lines 34-41; column 11, 45-55).

Brandt teaches gene therapy vectors which can be used to deliver therapeutic genes for gene therapy, and specifically teaches an HSV vector as well as an adenoviral vector and adeno-associated vector for use in gene therapy of the eye wherein the vector can be delivered to the eye by intravitreally injecting the vector which would necessarily encompass sub-retinal as well as intraocular delivery (e.g., see abstract, column 5, lines 5-20, column 8, lines 57-65, and column 9 lines 15-20).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time of filing to modify the teaching of Leboulch such that the bovine immunodeficiency viral vector taught by Poeschla (which is a lentiviral vector) is used to deliver and express the therapeutic gene and to deliver the lentiviral/BIV vector by intravitreally, subretinally or intraocularly injecting the gene therapy vector with a reasonable expectation of success.

The motivation to make such a modification is provided in part by Brandt who specifically teaches that adenoviral and AAV vectors can be used to treat eye disease by intravitreally, subretinally or intraocularly delivering the therapeutic vector; and in part by

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Poeschla who teaches that the BIV vector is better for transfecting difficult to target non-dividing cells of the nervous system including eye cells.

Response to Arguments and Declaration

3. Applicant's arguments filed 5/22/07 have been fully considered but they are not persuasive.

Applicants argue that WO99/264890 is not enabled for ocular gene therapy with endostatin. Applicants also assert that two patents cited by the Examiner as evidence that endostatin was an anti-angiogenic factor (U.S. Patent No. 5,827,702 and 6,206,104) are also not enabled (see page 9 of Applicants' response). Applicants refer to the Declaration of Dr. Guo which asserts that much of the original research on endostatin of the Folkman laboratory could not be repeated. Applicants, as well as the Declaration of Dr. Guo, refer to a number of prior and post filing reference to support the position that there is a lack the references cited by the Examiner are not enabled. Applicants also cite *Boehringer Ingelheim Vetmedica, Inc. v. Schering-Plough Corp. and Schering Corp.*, 320 F.3d 1339, 65 USPQ2d 1961 (Fed. Cir. 2003) as well as *Beckman Instruments, Inc. v. LKB Produkter AB*, 892 F.2d 1547, 1551, 13 USPQ 2d 1301, 1304 (Fed. Cir. 1989).

Applicants arguments, the Declaration of Dr. Guo and the court cases cited by Applicants have been fully considered, but re not persuasive.

First, it is noted that in a recent decision, *Rasmusson v. SmithKline Beecham Corp.*, 75 USPQ2d 1297 (Fed. Cir. 2005), the court indicated:

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The standard for what constitutes proper enablement of a prior art reference for purposes of anticipation under section 102, however, differs from the enablement standard under section 112. In *In re Hafner*, 410 F.2d 1403 [161 USPQ 783] (CCPA 1969), the court stated that “a disclosure lacking a teaching of how to use a fully disclosed compound for a specific, substantial utility or of how to use for such purpose a compound produced by a fully disclosed process is, under the present state of the law, entirely adequate to anticipate a claim to either the product or the process and, at the same time, entirely inadequate to support the allowance of such a claim.” *Id.* at 1405; see *Schoenwald*, 964 F.2d at 1124; *In re Samour*, 571 F.2d 559, 563-64 [197 USPQ 1] (CCPA 1978). The reason is that section 112 “provides that the specification must enable one skilled in the art to ‘use’ the invention whereas [section] 102 makes no such requirement as to an anticipatory disclosure.” *Hafner*, 410 F.2d at 1405; see 1 Donald S. Chisum, *Chisum on Patents* §3.04[1][c] (2002); see also *In re Cruciferous Sprout Litig.*, 301 F.3d 1343, 1349-52 [64 USPQ2d 1202] (Fed. Cir. 2001) (finding anticipation where applicant sought a patent based on a new use for a previously disclosed method).

Since *Hafner*, this court has continued to recognize that a prior art reference need not demonstrate utility in order to serve as an anticipating reference under section 102. See *Schoenwald*, 964 F.2d at 1124 (“it is beyond argument that no utility need be disclosed for a reference to be anticipatory of a claim”); *In re Donohue*, 632 F.2d 123, 126 n.6 [207 USPQ 196] (CCPA 1980) (“proof of utility is not a prerequisite to availability of a prior art reference under 35 U.S.C. §102(b)”), citing *In re Samour*, 571 F.2d at 563-64; see also *Application of Lukach*, 442 F.2d 967, 969 [169 USPQ 795] (CCPA 1971) (recognizing that there are “anomalies between the requirements for claim-anticipating disclosures and for claim-supporting disclosures” and citing *Hafner* as an example).

Also, see *Impax Laboratories Inc. v. Aventis Pharmaceuticals Inc.*, 81 USPQ2d 1001 (Fed. Cir. 2006).

Therefore, in view of *Rasmusson* and *Impax*, WO99/26480 provides sufficient teaching to anticipate the indicated claims, regardless of the lack of working examples and any problems repeating the endostatin experiments of the Folkman lab.

Furthermore, based on the totality of the art at the time of filing, one of ordinary skill in the would accept endostatin as an anti-angiogenic molecule. For instance, U.S. Patent No. 5,854,205 and 6,174,861 (previously cited) as well as Sauter et al. (PNAS 200, cited by

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Applicants in 8/30/02 IDS) and O'Reilly et al. (Cell Vol. 88:277-285, 1997) teach that endostatin is an anti-angiogenic factor which can be used to inhibit angiogenesis in a subject.

Additionally, the references cited by Applicants indicate, at best, that there is inconsistency in using endostatin as an anti-cancer agent. It is respectfully pointed out that at least some of the references cited by Applicants recognize endostatin as an anti-angiogenic factor.

For instance, Jouanneau et al. teach, "Here, we have evaluated the efficacy of one of the most promising natural inhibitors of angiogenesis described to date, endostatin, in a human neuroblastoma xenograft model in nude mice... The in vitro activity of soluble endostatin was confirmed on bovine capillary endothelial cells and human umbilical vein endothelial cells." (See abstract). Figure 2 of Jouanneau demonstrates the in vitro anti-angiogenic activity of endostatin.

Eisterer et al. teach, "A variety of studies have indicated endostatin to be a potent anti-angiogenic agent both in vitro and in vivo, and a human malignancy that might be sensitive to endostatin is human B-lineage acute lymphoblastic leukemia (B-ALL)." (See abstract) Although Eisterer does not teach that endostatin is an effective anti-cancer agent, there is no doubt that Eisterer recognizes the antiangiogenic activity of endostatin.

Also, Bachelot teaches:

"One of the most promising of these recently described natural inhibitors of angiogenesis is endostatin, a C-terminal fragment of collagen XVIII. In-vitro, endostatin strongly inhibits endothelial cell proliferation and migration. Initial in-vivo studies were impressive, recombinant endostatin was shown to induce regression and prevent the growth of experimental tumors in mice. Several studies by independent teams were published thereafter; they either described different forms of the recombinant protein, or developed gene therapy approaches. Most groups have shown perceptible activity in

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mouse tumor models, albeit without evidence of tumor regression." (Emphasis added, See abstract).

Therefore, Bachelot teaches that most studies have shown perceptible endostatin antiangiogenic activity in mouse tumor models even if evidence of tumor regression was not seen.

Therefore, given the strong evidence in the prior art indicating that endostatin is an antiangiogenic protein and considering that methods of gene therapy of the eye were taught in the prior art, the conclusion is inescapable that, at the time the instant application was filed, Leboulch et al. was enabled for treating ocular neovascularization of the eye by delivering to the eye a gene therapy vector that encodes and expresses endostatin.

Therefore, Applicants arguments are not persuasive and the rejections are maintained.

Conclusion

4. **No claim is allowed.**

5. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event,

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however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to J. Eric Angell whose telephone number is 571-272-0756. The examiner can normally be reached on Monday-Thursday 8:00 a.m.-6:00 p.m..

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James Douglas Schultz can be reached on 571-272-0763. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/J. E. Angell/
Primary Examiner
Art Unit 1635